Journal of Thermal Biology 78 (2018) 131–139

Contents lists available at ScienceDirect

Journal of Thermal Biology

journal homepage: www.elsevier.com/locate/jtherbio

Heat stress in poultry production: Mitigation strategies to overcome the future challenges facing the global poultry industry

Aamir Nawab[a], Fahar Ibtisham[a], Guanghui Li[a], Barbara Kieser[b], Jiang Wu[a], Wenchao Liu[a], Yi Zhao[a], Yasir Nawab[c], Kongquan Li[d], Mei Xiao[a], Lilong An[a,*]

[a] Department of Livestock Productions and Management, Agricultural College, Guangdong Ocean University, Zhanjiang 524088, Guangdong, China
[b] Department of Foreign Language, Guangdong Ocean University, Zhanjiang 524088, Guangdong, China
[c] Faculty of Veterinary Medicine, PMAS-Arid Agriculture University Rawalpindi, Pakistan
[d] Marine Engineering and Navigation College, Guangdong Ocean University, Zhanjiang, Guangdong, China

ARTICLE INFO

Keywords:
Poultry farming
Climate change
Heat stress
Productivity
Food safety
Mitigation

ABSTRACT

Worldwide, the effect of climatic variations has become a great challenge in poultry production. As global climate is changing, it alters the environmental temperatures, precipitation patterns and atmospheric carbon dioxide. Poultry farming mainly depends on climatic conditions such as temperature and humidity. Several factors can be involved but heat stress is one of most important environmental factor influencing a wide range of chickens performances including reduced feed intake which, in turn, affects growth rate, body weight, meat quality, egg quality, egg production, semen quality and fertility; these negative influences result in great economic losses. Heat stress associated food safety issues have gained special importance due to public awareness and an abundance of available scientific information. Environmental modifications (early heat conditioning, open sheds and cooling systems) and nutritional strategies (early feed restriction, electrolyte, vitamin and mineral balance) cannot satisfy the special needs of stressed poultry. Therefore, there exists a crucial need to explore effective strategies including genetic markers to enhance thermo-tolerance and productivity of poultry birds in hot regions of the world.

1. Introduction

The poultry industry has gained significance all over the world. In 2012, the FAO (Food and Agriculture Organization) estimated 103.5 million tons of annual global chicken meat production which contributed about 34.3% to global meat production (Pawar et al., 2016). Among food animals, chicken meat and eggs are the most efficient protein sources. Overall, poultry farming has performed a leading role in the livestock sector in some parts of the world (Sebho, 2016). Chickens are providing economical, healthier food than red meat and other protein sources (Leinonen et al., 2014).

Animal's farming system is affected by several climatic factors but environmental stress has gained special attention in livestock especially in the poultry farming, due to public awareness and an abundance of available scientific information (Lara and Rostagno, 2013). Variations of environmental factors such as sun light, temperature, humidity and characteristics of animal metabolism and the mechanism of thermoregulation can cause imbalances in the animal body (Leinonen et al., 2014). An increase in temperature might influence the susceptibility of pathogens (bacteria and parasites) in the environment of chickens. Heat stress negatively effects the plant growth (cereal grains) and causes poor feed quality which can affect the poultry growth rate (daily weight gain) due to reduce feed efficiency (Gupta et al., 2016; Ibtisham et al., 2017a). Ayo et al. (2011) described that layer chickens had a 20% reduced feed intake during hot and humid weather. The above authors recorded a decrease in egg production and a drop in hen-day production.

Heat stress results in annual economic losses of $128 to $165 million in the poultry industry alone. The total annual economic loss is $1.69 to $2.36 billion in the U.S. livestock industry (Lara and Rostagno, 2013). High environmental temperature is injurious to chickens which causes high rate of morbidity and mortality; thereby hazardous to human nutrition (Nienabar, 2007; Renaudeau et al., 2012; Sebho, 2016). Several studies have reported that under thermal conditions, the feed conversion ratio (FCR) increases more in fast growing broilers than in slow growing layers influencing adversely the production cost (Loyau et al., 2013).

Modern commercial poultry produces more body heat due to their

* Correspondence to: Haida Road, Mazhang District, Zhanjiang 524088, Guangdong, China.
E-mail address: anlilong@126.com (L. An).

https://doi.org/10.1016/j.jtherbio.2018.08.010
Received 30 March 2018; Received in revised form 16 June 2018; Accepted 18 August 2018
Available online 22 August 2018
0306-4565/

fast metabolism. This makes birds more sensitive to environmental temperature (Settar et al., 1999; Deeb and Cahaner, 2002; Fisinin and Kavtarashvili, 2015; Pawar et al., 2016). An elevated climatic temperature effects the behavioral, physiological and immunological response of broilers and layers causing unfavorable consequences like immune-suppression, endocrine disorder and electrolyte imbalance which reduces the profitability (Mazzi et al., 2003; Quinteiro-Filho et al., 2012; Lara and Rostagno, 2013). Therefore, there is a crucial need to study the effects of heat stress on poultry production, in order to develop various effective mitigation strategies to reduce consequential production loss as well as aiming at developing heat resistant poultry breeds for successful poultry production in hot regions of the world.

2. Stress what is it…?

Stress is an interesting topic for researchers. The word stress has no universal definition. Stress refers to any condition that affects the biological mechanisms of the body - human or animal (Virden and Kidd, 2009; Cirule et al., 2012). Another definition of stress states that it is a stimulus that produces a threat to the body at any time. Stress alters the internal body temperature. When a stressor adversely impacts the animal physiology, this condition is known as distress (Moberg, 2000; Virden and Kidd, 2009). Stress can be classified as specific and/or nonspecific (Siegel, 1980; Virden and Kidd, 2009). Short-term stress is usually induced by a specific stressor such as variation in environmental temperature (Siegel, 1980; Virden and Kidd, 2009). When an animal takes measures to accommodate the stressor then it is called a nonspecific stress (Siegel, 1980; Virden and Kidd, 2009). In addition, stress has three stages comprising alarm, resistance and exhaustion. i) If the threat or stressor is identified then it is called state of alarm. ii) If stimulus or stressor remains for long time, then the body tries to adapt the environment by developing resistance (Virden and Kidd, 2009). iii) If all resistance resources are deficient and the body is unable to stabilize its normal physiology this is referred to as exhaustion stage. Various factors affect the animal health but heat stress is a critical form amongst the environmental stressors which adversely affects the entire cycle of a bird's production.

3. Mechanism of heat stress

Generally, poultry birds express different types of responses according to intensity and duration of heat stress. A study reported that discomfort zone causes heat stress and birds spend less time in feeding, moving, walking and more time in panting, drinking, elevating wings and resting. The neuroendocrine system performs the central role in the maintenance of healthy body functions in humans as well as in animals. High temperatures alter the neuroendocrine system (Fig. 1) and activate the sympathetic-adrenal medullar axis (SAM) including the hypothalamic-pituitary-adrenal (HPA) axis (Shini et al., 2008) which increase the glucose synthesis for the survival of chickens during stressed condition (Katarzyna and Sembratowicz, 2012). The HPA axis is the main stressor marker (Cockrem, 2007) rather than corticosterone, because corticosterone concentration can be increased under positive or negative stimuli due to the presence of a mate or an invader. The SAM also controls the fight or flight response, because it detects response and transfers stimuli from the hypothalamus to the adrenalin gland (Smith and Vale, 2006). Under stressful conditions, epinephrine (adrenalin) is secreted from SAM, which initiates fast response in the form of tachycardia (increased heart rate) and glucose synthesis (Dickens et al., 2010). It is difficult to evaluate stress when epinephrine is secreted at a high rate during stress conditions.

Cortisol and corticosterone are primary glucocorticoids (Manuja et al., 2012). Corticosterone is present in birds and rodents, while cortisol is exposed in cattle, pigs, sheep and fish (Mormede et al., 2007). Conversely, corticosterone is secreted from the HPA axis and pituitary gland (adreno-cortico-trophic hormone) (Mormede et al., 2007; Romero et al., 2015). Corticosterone secretion is slower than adrenalin, and has a more persistent effect (Mormede et al., 2007), therefore the corticosterone level is considered as a good indicator of stress. In addition, several reports showed that long time secretion of corticosterone under chronic stress may have detrimental effects on chickens, such as depression, reduced immunity, cardiovascular problems, and breakdown of muscle due to gluconeogenesis and decreased cognition (Romero et al., 2015).

Another possible mechanism mentioned is that heat stress induces infertility in farm animals as well as in domestic chickens. High-temperature environmental stress impairs the secretion of gonadotropin-releasing hormone (FSH and LH) in laying birds which, in turn, disturbs the reproductive performance (Dantzer and Keith, 1989; Ayo et al., 2011). Dantzer and Keith (1989) determined that heat stress (environmental, physically and mentally etc.) causes distress in the birds and affects the immune response of chickens through initiation of inflammatory cytokine (interlukin-1 etc.). This constrains the function of the hypothalamic-pituitary-gonadal axis and reduces the defense system in poultry.

4. Effect of heat stress on physiological response

High temperature affects the physiological functions of poultry birds at any stage of life which in results affects the poultry production performance. The physiological behavior influences the growth rate and the production costs (Neves et al., 2010, 2014). Day old chicks have a fast metabolism and growth rate, thus they have poor ability to adjust the environment fluctuations. Chicks don't have sweat glands; thereby they are highly sensitive to heat stress. First neurogenic system is encountered under a stressor. Neurogenic system increases blood sugar, respiration, muscle tone and nerve sensitivity. Epinephrine and norepinephrine are secreted by the activation of the neurogenic system. Heat stress affects the hypothalamic-pituitary-adrenal cortical system and produces a corticotrophins-releasing factor from the hypothalamus, which sends a message to the pituitary to release an adrenocorticotropic hormone (ACTH) (Lotvedt et al., 2017). ACTH is released from the pituitary while corticosteroid is secreted from the adrenal cortical tissue. Elevated levels of corticosteroids in circulation alter the glucose synthesis, the mineral metabolism and are responsible for hypercholesterolemia, cardiovascular diseases, gastrointestinal lesions, and variations in immune functions (Binsiya et al., 2017).

Animals maintain homeostasis under high temperature condition via conduction, convection, and evaporative heat loss by vasodilatation and perspiration (Pawar et al., 2016). Primarily, stress changes the metabolic function of chickens and induces the production of glucose for maintenance of homeostasis during the presence of stressors. Under high temperature, air sacs play a key role in the gaseous exchange as they increase the air circulation toward the surface. As a consequence evaporation causes dissipation of heat (John, 2009). However, it is worth mentioning that increased panting exhales more carbon dioxide and induces higher blood pH (respiratory alkalosis) (Borges et al., 2004). This disturbs the bicarbonate and free calcium availability in circulation for the mineralization of the egg shell. This phenomenon is significant in the layer industry that needs to avoid poor egg production (El-Tarabany, 2016).

5. Effect of heat stress on production performance

Several studies in poultry have investigated that high temperature influences the health, physiology and efficiency of chickens (Fig. 2) (Deeb et al., 2002; Ghazi et al., 2012; Attia et al., 2017b). Sohail et al. (2012) reported that broilers at 42 days of age exposed to chronic heat exposure had a 16.4% reduced feed intake, 32.6% body weight and a 25.6% higher feed consumption ratio. Chronic heat stress in broilers negatively affects fat metabolism, muscle growth and reduces the meat quality and chemical profile due to electrolyte imbalance and activation

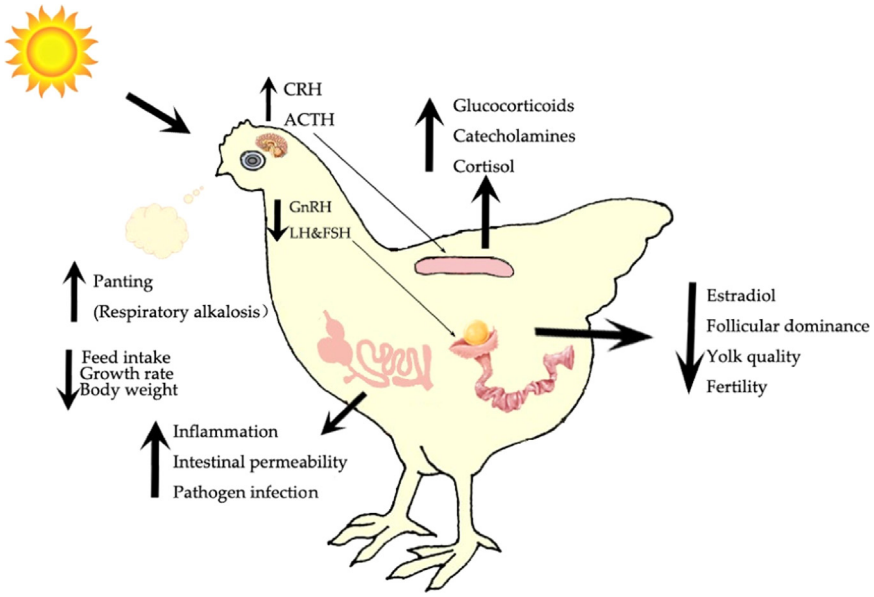

Fig. 1. Mechanism of heat stress. CRH, corticotropin-releasing hormone; ACTH, adrenocorticotropic hormone; GnRH, gonadotropin-releasing hormone; LH, luteinizing hormone; FSH, follicle-stimulating hormone.

of lipid peroxidation (Babinszky et al., 2011; Dai et al., 2012; Sokołowicz et al., 2016; Kim et al., 2017). Furthermore, heat stress reduces protein content and increases fat deposits in chickens (Ayo et al., 2011).

High ambient temperature has been associated with higher mortality and welfare issues during transportation (Vecerek et al., 2016). The death during transportation is generally associated with chickens weight (higher weight causes higher mortality (Caffrey et al., 2017). Under harsh environmental conditions, feed intake had reduced 28.58 g/chicken/day and 28.8% egg production in laying hens in a 12-day trial (Zhang et al., 2017). Overall, heat stress reduced whole egg weight, egg shell thickness, only eggshell weight and eggshell by percent 3.24%, 1.2%, 9.93%, 0.66%, respectively (Ebeid et al., 2012; Mack et al., 2013). The resulting variations may be explained due to stress duration, intensity as well as age of birds, physiological status and genetic potential.

6. Effect of heat stress on reproductive profile

Reproductive efficiency is a key factor affecting profitability in many livestock production systems (Ibtisham et al., 2018). Environmental stress suppresses the reproductive efficiency of birds by reducing the size of reproductive organs (follicular and oocyte development). Under high temperatures, heat stress decreases the secretion of the gonadotrophin-releasing hormone (GnRH) which ultimately reduces the concentration of follicle-stimulating and luteinising hormones

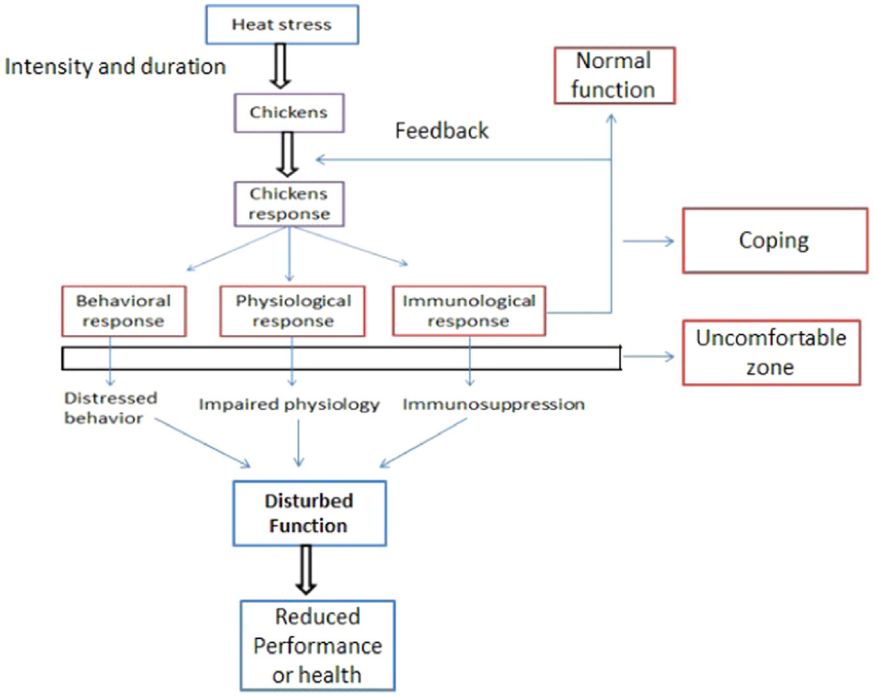

Fig. 2. Poultry responses to heat stress.

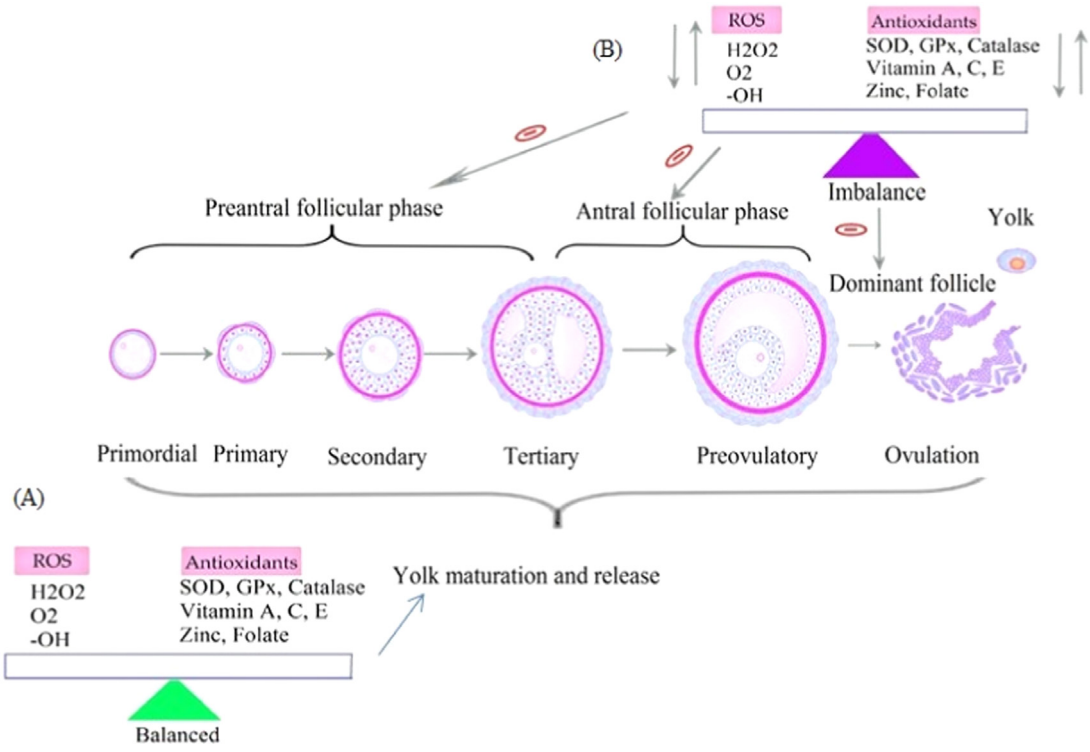

Fig. 3. Effects of heat stress on follicular growth during process of ovulation. (A) Reactive oxygen species (ROS) and anti-oxidants balance is required for follicular maturation, atresia, and the growth of the dominant follicle (B) Heat stress induces imbalance in ROS and anti-oxidant generation and lead to ovulatory dysfunction. GPx, glutathione peroxidase; H_2O_2, hydrogen peroxide; O_2, superoxide; SOD, superoxide dismutase; OH, hydroxyl radical.

in laying birds and brings infertility (Ayo et al., 2011). Climatic variations delay the process of ovulation via reducing follicular size (Kala et al., 2017), estradiol concentrations and expression of LH receptors (Kala et al., 2017). Specifically, heat exposure increases the reactive oxidative species (ROS) in layer birds and reduces the yolk quality by inducing lipid peroxidation of cytome-membranes (Fig. 3) (Papadopoulou et al., 2017). The yolk maturation rate may be reduced due to the deviation of the fatty acid composition, heat shock proteins, and antioxidants level in hot climates (Ayo et al., 2011). It is well-known that temperature, pH, ion concentration, and other environmental factors affect all phases of semen production, sperm metabolism, quality and motility (Darszon et al., 1999; Riaz et al., 2004). However, it is not yet clear that heat exposure is responsible for poor semen quality and fertility.

It is well-known that some of the ion channels regulating ion exchange during the initial phases of epithelial cell differentiation end up in mature spermatozoa, responsible for their physiological properties (Santi et al., 2013). Therefore, it is concluded that stress has detrimental effects on testicular function through inhibition of intracellular ion exchange (Bonato et al., 2014; Gallo and Tosti, 2015). The reproductive efficiency of a broiler breeder rooster is significantly depressed during high environmental temperatures (Ameen et al., 2014). Bonato et al. (2014) described that the broiler breeders are more prone to heat associated infertility than the females. When male broiler breeders exposed to 32 °C temperature, male fertility were declined by 42% and in vivo sperm-egg penetration were decreased to 52%, compared to the ratio of males that were maintained at 21 °C (Ayo et al., 2011). Very clearly, this observation demonstrates that spermatozoa motility was affected due to depreciating the qualitative and quantitative seminal characteristics and thus resulted in infertility (Ameen et al., 2014). Continuous exposure of poultry to the heat stress impairs embryonic growth and development permanently (Noiva et al., 2014) and produces teratogenic (birth) defects in chicks.

7. Effect of heat stress on gut health

Gut health plays a significant role in the efficient digestion and absorption of feed, water and electrolyte balance as well as in immune system development (Jahejo et al., 2016). The gut acts as a barrier eliminating toxins and infectious agents. Under climatic variations, several types of bacterial pathogens inhabit the gut and disturb its ecosystem. The gut microbial ecosystem is influenced by various factors including feed composition, feed additives (probiotic, prebiotic, organic acids, feed enzyme etc.), feeding practices, environmental stress, genetics and temperature in animal housing (Tuohy et al., 2003; Zoetendal et al., 2004). These factors have potential effects on gut health and microbiota. Today's poultry seems to be susceptible to high environmental temperatures. Gut health is compromised after heat exposure which results in reduced nutrient absorption, gut wall integrity and immune system dysfunction. All of these cause poor performance, increased disease susceptibility and higher mortality in chickens (Varasteh et al., 2015). Under thermo-neutral environment, the gut has the ability to efficiently digest and absorb the nutrients through trans-cellular transport via specific receptors. These gut epithelial cells are tightly connected via intercellular junction complexes (Fig. 4). These complexes are a significant component of the intestinal barrier and stabilize the integrity of the epithelial barrier (Kamada et al., 2013; Goulet, 2015).

Gut epithelia junction complexes consist of adherent junctions (AJ), tight junction (TJ), gap junctions (GJ) and desmosomes (Fan et al., 2015). Desmosomes are associated to keratin filaments whereas adherent junctions (AJ) are located beneath the tight junction (TJ) and are involved in intracellular communication (Perez-Moreno and Fuchs, 2006; Tellez et al., 2017). Both junction such as TJ and AJ (apical junctional complex) are connected to the actin cytoskeleton (Salvo-romero et al., 2015; Hu et al., 2013). Desmosomes and gap junctions (GJ) are involved in cell adhesion and intracellular signaling respectively (Tellez et al., 2017). The cytoskeleton (protein complex) maintains the

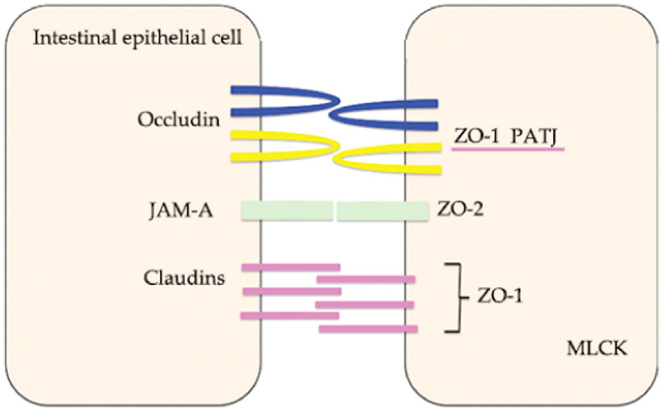

Fig. 4. Tight Junction proteins stabilize intestinal integrity. Several classes of tight junction proteins (Claudins, JAM-A, PATJ and ZO-1/2) play a vital in cell permeability. Heat stress affects these TJ and reduces the gut integrity. JAM-A, junctional adhesion molecule-A; PATJ, PALS1-associated TJ protein; ZO-1/2, zonula occludens 1/2; MLCK, myosin light kinase.

cell structure of all eukaryote. Damaged cytoskeleton decreases the intestinal integrity (Siddiqui et al., 2015).

TJ is a complex protein structure that forms trans-membrane protein channels across the epithelium and allows the transport of different substances. TJ is regulated through signaling pathways and interacts with trans-membrane proteins and the actomyosin ring is organized by several intracellular proteins, comprising myosin light kinase (MLCK), mitogen-activated protein kinases (MAPK), protein kinase C (PKC), PATJ (PALS1-associated TJ protein) and the Rho family of small GTPases (Tellez et al., 2017). Occludin phosphorylation is associated with disruption of TJ. Therefore, the occludin protein is crucial in the regulation of TJ barrier functions (Siddiqui et al., 2015). During high-temperature environmental conditions, the TJ barrier is compromised and luminal contents enter into the blood circulation. Therefore, leaky gut (Guy and Vincent, 2018) induces chronic systemic inflammation which reduces the disease-resistance capacity of birds (Tellez et al., 2017).

8. Effect of heat stress on immune response

The rising ambient temperature impairs performance as well as immune status of poultry birds (Tirawattanawanich et al., 2011). Environmental stress reduces chickens thymus, spleen, bursa of fabricus, liver and lymphoid organ weights and causes immune-suppression. A study has observed that broilers exposed to thermal stress had a decreased ratio of circulating antibodies, IgG and IgM, and reduced systemic humoral responses (Aengwanich, 2008; Lara and Rostagno, 2013; Alagawany et al., 2017).

Heat stress reduces the intraepithelial lymphocytes, IgA-secreting cells (intestinal tract) and antibody titer in laying hens as well as affect the macrophages performance of phagocytosis in broilers (Lara and Rostagno, 2013; Sugiharto et al., 2017). Moreover, many studies have described the heat stress reduces the phagocytic ability of macrophages and reduces macrophage basal and oxidative burst in broilers (Gomes et al., 2014). In addition, high temperature can alter the ratio of circulating cells and increase the ratio of heterophil to lymphocyte, due to lower lymphocytes and higher number of heterophils in circulation (Prieto and Campo, 2010; Lara and Rostagno, 2013). The poultry birds struggle to compensate thermal homeostasis, but stress conditions enhance the reactive oxygen species (ROS) production. TLR4 signaling pathway is activated especially during heat stress (Fig. 5). Toll-like receptors (TLRs) are a group of trans-membrane receptors that safeguard the host pathogen boundaries by recognizing microbial patterns (Huang, 2017). Bacteria are sensed by TLR2, TLR4/MD-2, TLR5, and TLR9 that detect lipoproteins, lipopolysaccharide, flagellin, and DNA,

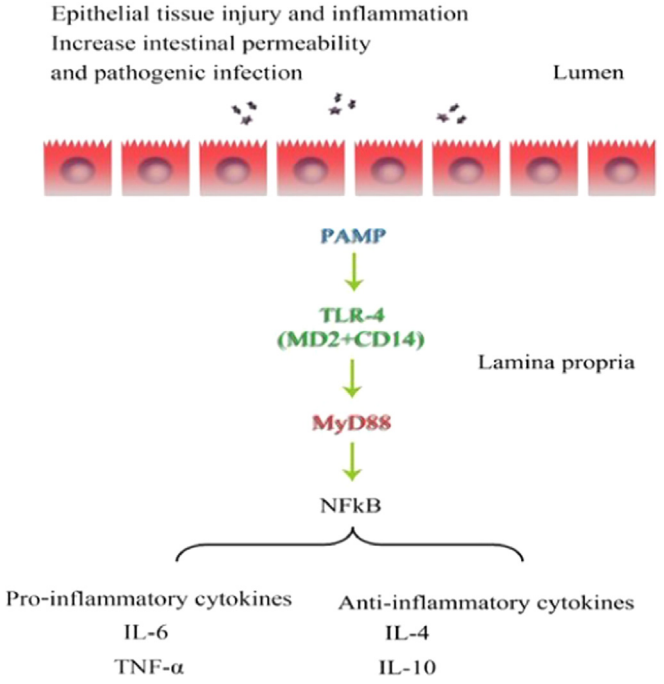

Fig. 5. Heat stress damage intestinal epithelium and reduce gut integrity. Pathogen associated molecular pattern (PAMP) are recognized by TLR4 + MD2 + CD14 complex after that MyD88 and nuclear transcription factors (NF-κB) is activated which results in activation of pro-inflammatory cytokines. TLR4, toll-like receptor 4; MD2, myeloid differentiation 2; CD14, cluster of differentiation 14; MyD88, myeloid differentiation primary response 88; TNF-α, tumor like necrosis factor-α; ILs, interleukins.

respectively (De Zoete et al., 2010). After TLR activation, a signaling cascade involving the adapter proteins myloid differentiation 88 (MyD88) (all TLRs except TLR3) results in the activation of nuclear transcription factors (NF-κB), which induce the pro-inflammatory cytokines and suppress immunity (Karnati et al., 2015).

9. Effect of heat stress on food safety

Food safety is considered a significant part of the modern food quality concept. Worldwide, poultry farming has to face the issue of food safety due to environmental stressful conditions. Several scientific studies have investigated that stress can adversely affect food safety via several possible mechanisms (Pawar et al., 2016). A report suggests that the shedding of pathogens in farm animals is due to stress conditions, but the underlying phenomenon is not yet fully elucidated (De Passille and Rushen, 2005; Lara and Rostagno, 2013; Pawar et al., 2016). Heat stress produces undesirable meat characteristics and quality loss in broilers (Kim et al., 2017). In addition, meat quality losses were also described during the transportation of broiler chickens from farms to processing units under hot climatic conditions (Dadgar et al., 2010). Heat stress has also been associated with low egg production and egg quality in laying hens (Bozkurt et al., 2012; Pawar et al., 2016).

10. Mitigation strategies to reduce heat stress in poultry

10.1. Environmental strategies

One of the key factors to reduce the effects of heat stress is environmental modification. However, environmental technical strategies cannot alleviate heat stress in the poultry farm if nutritional programs, disease control and genetic status of poultry are not optimal. Stressful environmental condition can be reduced by using basic designing rules to enhance poultry farming in a hot environment. For instance, the

shape of housing (semi-open buildings); ventilation (air movement into and out of buildings to remove ammonia, carbon dioxide and moisture) in hot and humid locations; natural or artificial shading (surrounding vegetation and grass cover) and water consumption (Lin et al., 2006; Butcher and Richard, 2012; Fisinin and Kavtarashvili, 2015; Pawar et al., 2016). Another factor that can avoid heat built-up is the condition of the roofing. Roofs should be clean, rust and dust free. A shiny surface reflects solar radiation more than a dark or rusty roof. Roof reflectivity can be increased by painting with metallic zinc paint or by an aluminum roof (Pawar et al., 2016). Proper ventilation can increase convective cooling by using circulation fans.

Heat production and body heat loss in poultry are both related to heat stress. Heat stress should be managed in poultry housing to reduce the unfavorable consequences on chickens. Stress-associated health problems can be minimized via ventilation technologies (Pawar et al., 2016). Ventilation equipment should be installed properly and maintained regularly. Additional ventilation fans and generators should be available in order to manage an emergency situation (Kapetanov et al., 2015). According to 1994 regulations for the welfare of livestock, the installation of an alarm system that alerts to the failure of the ventilation system is required; which might protect chickens from uncomfortable or stressful conditions, especially during periods of hot weather (Kapetanov et al., 2015). Therefore, emergency equipment (fail-safe electric panels) should be designed to prevent temperatures rising in poultry housing more than 5 °C above the outside temperature.

Under condition of high temperature or humidity, catching, loading and transporting cause particular stress risk to the birds. Thus, plans including daily attention to metrological forecasts should be made in advance to take the correct actions in order to reduce the risk of overheating. Catching and loading of chickens should be done early in the morning. Ventilation fans or additional mobile fans should be provided for the yet uncaught birds during the catching and loading operations. Water should be regularly offered to the uncaught birds by lowering the drinkers from time to time, thus providing adequate water supply, feed and light for the birds. Density is one of the critical factors during the hot summer season (Kapetanov et al., 2015). Therefore, the number of birds should be adjusted according to crate design and environmental temperature. Chickens crates should be settled on the vehicle thus allowing for sufficient ventilation. Ventilation holes on fixed crates must be opened. Bird crates and/or fully loaded vehicles should be parked in well-ventilated lairs. The evaporative heat dissipation depends on temperature and humidity as it increases with temperature and decreases with increasing humidity (Lin et al., 2006; Lara and Rostagno, 2013; Pawar et al., 2016). Thus, high temperature along with high humidity is more injurious to broilers than high temperature with low humidity. On the other hand, early heat conditioning (EHC) is one of the promising methods of managing the effect of heat stress in broiler chickens. EHC refers to the process of high temperature (36 °C) exposure to broiler chicks for 24 h at age of 3–5 d (Lin et al., 2006; Pawar et al., 2016). Furthermore, another study suggested that poultry eggs exposed to high temperature during incubation may increase the thermo-tolerance throughout a chicks life (Fisinin and Kavtarashvili, 2015). These possible strategies may help to reduce the stress load in poultry.

10.2. Feeding strategies

Several successful dietary strategies have been used to mitigate the harmful effects of increasing environmental temperature. The purpose is to fulfill the special needs of chickens in hot climates via application of water, nutrients, electrolytes, vitamins and minerals. It has been noticed that management, such as restriction of feed (Dale and Fuller, 1979; Daghir and Lebanon, 2009; Attia et al., 2017a), fat addition and excess protein reduction (Rahman et al., 2002; Ghazalah et al., 2008) are specially recommended to minimize the detrimental effects of heat stress and improve the birds performance (Daghir and Lebanon, 2009; Attia et al., 2017b; Kumari et al., 2018). Early feed restriction (EFR) reduces the negative influence of heat stress on growth performance and immune system of broilers (Zulkifli et al., 1994; Khajavi et al., 2003; Lin et al., 2006; Renaudeau et al., 2012; Kumari et al., 2018). A study has reported that feed restriction (about 60%) to chicks on day 4, 5 and 6 increased growth and the survival rate in response to exposure of heat stress on days 35–41 (marketing age) (Lin et al., 2006). Research in Russia has suggested that 5% fat and 4% soybean or palm oil addition in the diet of broiler chicks had improved chickens production (Fisinin and Kavtarashvili, 2015). Fat supplementation in feed also increases the nutrient value of other feed components and reduces the feed retention time in the intestinal tract and enhances the utilization of nutrients (Mateos et al., 1982; Pawar et al., 2016). During extreme stressful conditions, the feed should be composed of highly digestible nutrients (Kapetanov et al., 2015). Therefore, the poultry industry has suggested the use of quality protein and amino acids (methionine and lysine etc.) in order to reduce the heat increment and the negative effects of high temperature (Daghir and Lebanon, 2009; Attia et al., 2017b; Kumari et al., 2018).

Under heat stress, minerals (Fe, Zn, Se and Cr) and vitamins (vitamin A, C and E) are excreted from the body of chickens resulting in mineral and vitamin deficiency (Sahin et al., 2009; Pawar et al., 2016; Kumari et al., 2018). The dietary supplementation of vitamins, minerals and electrolyte balance has been reported to reduce mortality and improve the growth rate of poultry under harsh environmental condition (Fisinin and Kavtarashvili, 2015). The dietary addition of salts electrolytes ammonium chloride (NH4C), potassium chloride (KCl), and sodium bicarbonate ($NaHCO_3$) in feed or drinking water improve the performance of broilers and protect them from the injurious effects of heat exposure (Ahmad et al., 2008; Renaudeau et al., 2012; Fisinin and Kavtarashvili, 2015; Yosi et al., 2017). The addition of sodium bicarbonate 4–10 kg/t feed helps to maintain the acid-base balance in the birds resulting from panting (respiratory alkalosis) (Fisinin and Kavtarashvili, 2015). Potassium chloride 0.25–0.5% or 0.5–1.0% in drinking water and/or feed restores the balance of electrolyte salts (Fisinin and Kavtarashvili, 2015). Several nutritionists suggest that the administration of Vitamin A (8000 IU/kg) in the feed may overcome the detrimental effects of stressed laying hens (Lin et al., 2002; Daghir and Lebanon, 2009; Attia et al., 2017b; Kumari et al., 2018). A feeding experiment has shown that diet supplementation of vitamin E (200 mg/kg) had increased serum IgM and IgG level as well as improved phagocytic ability of macrophage as compared to control group (Fisinin and Kavtarashvili, 2015). Heat stress causes unfavorable changes in the gut microbiota. The supplementation of probiotic based *lactobacillus* strains may help to balance the gut microbiota of chickens suffering from high temperature (Lan et al., 2004; Lin et al., 2006; Pawar et al., 2016). Several special feed supplements like, dietary enzyme, baking soda, zinc bacitracin, osmo-protective supplements may also be helpful in reducing heat-stress mortality (Lara and Rostagno, 2013; Fisinin and Kavtarashvili, 2015).

The physical appearance and feed quality (crumb, pellets or mash) encourage appetite in birds. The feed industry has collected data and claims that consumption of pellet-feed requires one-third less time compared with spilled feed and allows chickens to save around 6% of energy (Fisinin and Kavtarashvili, 2015). This may controls the heat production. Feed storage should not be longer than two months; mainly during the hot season in order to reduce the chance of mycotoxin. Feeding time should be in the morning/evening or follow the night feeding principle. The feeding schedule in laying hens should comprise 1/3 of the ration given early in the morning and 2/3 late in the afternoon. An experiment has revealed that feeding at night-time can improve the eggshell quality (Kapetanov et al., 2015). The feed composition should be adjusted properly and formulated according to the special requirements of the poultry breed. During feeding low energy light or dimmers should be used to reduce the birds activity which ultimately reduces the heat load. Some authors recommend the limiting

feeding to broilers about 4–6 h prior to the daily peak in temperatures in order to reduce the increase of body temperature during feed intake in hot climates (Pawar et al., 2016).

10.3. Genetic selection strategies

Breed improvement programs have increased growth rate and feed efficiency in poultry but results in lower heat tolerance (Hoffmann, 2010; Pawar et al., 2016; Mideksa, 2017). However, the response of genotypes in high temperatures might be different from those in a thermo-neutral environment. Gwaza et al. (2017) reported that three chicken lines (broiler), showed similar performance at temperature 18 °C (autumn), however, variations in feed consumption, body weight and feed efficiency were observed at temperature 28 °C (summer). Heat stress reduces the disease resistance of birds due to loss of immunity (Shini et al., 2010; Nazar et al., 2017).

In poultry, there are several traits that favor heat tolerance. Feather features can be controlled by selection and/or mutations of dominant genes such as naked neck (Na) or frizzle (F) genes (Lin et al., 2006; Zerjal et al., 2013; Gwaza and Nachi, 2015; Pawar et al., 2016). The *Na* gene results in higher growth rate and meat yield in broiler chickens under thermo-neutral temperature; this effect is particularly noticeable at high temperatures. A high growth rate under high temperatures may be associated with a high concentration of triiodothyronine (T3) (Decuypere et al., 1993; Lin et al., 2006). It also reduces fat deposition in skin and breast muscles which increases heat dissipation via the neck (Raju et al., 2004).

A study has shown that the *Na* gene affects the feather growth directly and reduces the cover by 20% and 40% (relative to BW) in the heterozygous (*Na/na*) and homozygous (*Na/Na*) chickens group, respectively, as compared with the normal or fully feathered group under chronic heat stress (Deeb and Cahaner, 2001; Lin et al., 2006). In broiler chickens, the *Na/Na* or *Na/na* (naked neck) gene, compared with *na/na* (fully feathered) gene, improved feed efficiency and body weight but had lowered body temperature (Patra et al., 2002; Lin et al., 2006). Therefore, the Na gene in broiler must be considered especially in hot climates (Yalcin et al., 1997; Lin et al., 2006). Frizzle (F) feather gene reduces the feather insulation by curling and reducing the size of feathers (Lin et al., 2006; Abd El-Hack. et al., 2018). The positive effect of F gene on the growth rate of broiler is less than the effect of Na allele under high temperature (Lin et al., 2006; Pawar et al., 2016). However, there is an additive effect in the heterozygous gene utilization (Na/na F/f) in broiler chickens (Yunis and Cahaner, 1999; Fisinin and Kavtarashvili, 2015).

Furthermore, the sex-related recessive gene for dwarfism (dw) can be selected which reduces 30–40% of adult body size and metabolic heat production in broiler breeders (Lin et al., 2006; Pawar et al., 2016). However, the *dw* gene does not improve thermo-tolerance of broiler chickens under chronic heat stress (Deeb and Cahaner, 2001; Lin et al., 2006). Specifically, the scale-less mutation in chickens with fewer feathers is presently induced in a fast-growing line to improve thermo resistance (Cahaner et al., 2008). A study described that under thermal stress, the protein synthesis is mostly arrested, but heat shock proteins (highly conserved proteins) are rapidly synthesized and bind with heat-sensitive proteins to protect the degradation of proteins. Heat shock proteins also prevent proteins from precipitating and constantly affecting cell viability (Etches et al., 2008; Pawar et al., 2016).

Genomic selection plays a great role in livestock breeding to select the fittest animals (Ibtisham et al., 2017b). Genetic markers such as single nucleotide polymorphism (SNP) are currently a choice dealing with heat tolerance of poultry. These markers are essential to select chromosomal regions of poultry for heat stress tolerance (Yu et al., 2008; Kong et al., 2015; Pawar et al., 2016). Another potential technique in the livestock breeding industry is epigenetics. Epigenetics (gene expression, imprinting and silencing of transposons as well as modification of histone proteins, non-coding RNA, DNA sequencing and DNA methylation) may help to understand the mechanism of how environmental factors result in heritable variations in gene expression and/or genomic functions. This insight, in turn, can be useful to improve the birds heat tolerance while optimizing future poultry production (Scholtz et al., 2014; Triantaphyllopoulos et al., 2016). There is critical need to focus on the genotype of broilers to increase the world meat-chicken production especially in hot areas of the world (Renaudeau et al., 2012).

11. Conclusion and recommendation

Heat stress has become an emerging topic for many researchers due to global warming and food security issues. Heat stress negatively affects the physiological, immunological, reproductive and gut status of poultry birds because of poor heat tolerance of poultry genotypes, which causes huge economic losses in the poultry industry. Environmental modifications like adequate ventilation and cooling systems and nutritional adjustments may help to reduce metabolic heat production and maintain electrolyte balance in high-temperature stressful conditions. Thermo-tolerance can be enhanced by early-age heat conditioning and/or controlled fasting during the initial days of a chicks life to boost the survival rate of broilers under heat stress. However, these solutions have variable consequences depending on birds age, health status, breed, sex, management and geographical location. The availability of insufficient scientific data on genotypic characterization has critically damaged the opportunity of successfully competing with climate variations. FAO has predicted that 9.3 billion people may need to be fed in 2050 and about 11 billion in 2100 (Vos, 2015). Therefore, there is a severe need to investigate different mitigation strategies to overcome the future challenges to the poultry industry. Hence, genetic markers are being explored to identify specific genes in the selection of poultry breeds to increase heat tolerance and disease resistance. Epigenetics (early age embryo gene expression and imprinting) can also be an effective method to increase the heat tolerance of birds. Various cellular and molecular techniques such as RNA extraction, cDNA synthesis and quantitative PCR as well as examination of oxidative stress (malondialdehyde and thiobarbituric acid-reactive substances) and antioxidant enzyme activities (superoxide dismutase, glutathione peroxidase and catalase) can assist in understanding the various biological pathways involved in the physiological, reproductive and immunological responses of poultry birds to global warming. This may help to develop poultry breeds that perform better in hot climates. In addition, a specific breeding program should be planned to evaluate the potentiality of frizzled feather and naked neck genes under various environmental conditions to improve the poultry industry, particularly in the hot climatic regions of the world.

Acknowledgments

Special thanks to Journal of thermal biology for giving an opportunity to share knowledge on worldwide heat stress issues and strategies to overcome this problem. The author wish to thank beloved parents (Rana Nawab Ahmad and Mrs. Sultana), grandparents (Saith Bachal Din), uncle (Rana Maqbool Ahmad) and brother (Rana Kashif Nawab) for continued support and excellent mentorship. This work was supported by Science and Technology Planning Project of Guangdong Province, China (2010 B090400376).

Authors' contributions

The author wrote the main text of manuscript. The co-authors collected the data, contributed to the design and drafting of the paper. All authors read and approved the final manuscript.

Competing interests

The authors declare that they have no competing interests.

References

Abd El-Hack, M.E., Alagawany, M., Noreldin, A.E., 2018. Managerial and nutritional trends to mitigate heat stress risks in poultry farms. In: Part of The Handbook of Environmental Chemistry, Berlin, Heidelberg, pp. 1–14.

Aengwanich, W., 2008. Pathological changes and the effects of ascorbic acid on lesion scores of bursa of Fabricius in broilers under chronic heat stress. Res. J. Vet. Sci. 1, 62–66.

Ahmad, T., Khalid, T., Mushtaq, T., Mirza, M.A., Nadeem, A., Babar, M.E., Ahmad, G., 2008. Effect of potassium chloride supplementation in drinking water on broiler performance Under heat stress conditions. Poult. Sci. 87, 1276–1280.

Alagawany, M., Farag, M.R., Abd eL-Hack, M.E., Patra, A., 2017. Heat stress: effects on productive and reproductive performance of quail. World Poult. Sci. J. 73, 747–756.

Ameen, S.A., Opayemi, O.S., Ajayi, J.A., Adediwura, M.A., 2014. Evaluation of semen quality of five different Cockerel breed used in poultry industry in Nigeria. J. Environ. Issu. Agric. 6, 30–36.

Attia, Y.A., Al-harthi, M.A., Elnaggar, A.S., 2017a. Productive, physiological and Immunological responses of two broiler strains fed different dietary regimens and exposed to heat stress. Ital. J. Anim. Sci. 0, 1–13.

Attia, Y.A., Al-Harthi, M.A., Elnaggar, A.S., 2017b. Productive, physiological and Immunological responses of two broiler strains fed different dietary regimens and exposed to heat stress. Ital. J. Anim. Sci. 17, 1–12.

Ayo, J.O., Obidi, J.A., Rekwot, P.I., 2011. Effects of heat stress on the well-being, fertility, and hatchability of chickens in the Northern Guinea Savannah Zone of Nigeria: a Review. Vet. Sci. 2011, 1–11.

Babinszky, L., Veronika, H., Verstegen M.W.A., 2011. Impacts of climate change on animal production and quality of animal food products. In: Book: Climate Change - Socioeconomic Effects. Ch. 10. doi: ⟨http://dx.doi.org/10.5772/23840⟩.

Binsiya, T.K., Veerasamy, S., Bagath, M., Krishnan, G., Iqbal, H., Manimaran, A., Angela, L., Gaughan, J., Bhatta, R., 2017. Significance of hypothalamic-pituitary-adrenal axis to adapt to climate change in livestock. Int. Res. J. Agric. Food Sci. 2, 1–20.

Bonato, M., Irek, A.M., Paulina, K.R.T., Charlie, K.C., Schalk, W.P.C., 2014. Predicting Ejaculate quality and Libido in male Ostriches: Effect of season and age. Anim. Repro. Sci. 151, 49–55.

Borges, S.A., Fischer Silva, A.V., Majorka, A., Hooge, D.M., Cummings, K.R., 2004. Physiological responses of broiler chickens to heat stress and dietary electrolyte balance (Sodium Plus Potassium Minus Chloride, Milliequivalents Per Kilogram). Poult. Sci. 83, 1551–1558.

Bozkurt, M., Kucukyilmaz, K., Catli, A.U., Cinar, M., Bintas, E., Coven, F., 2012. Performance, egg quality, and immune response of laying hens fed diets supplemented with mannan-oligosaccharide or an essential oil mixture under moderate and hot environmental conditions. Poult. Sci. 91, 1379–1386.

Butcher, G.D., Richard M., 2012. Heat Stress Management in Broilers.VM65 Series of the Veterinary Medicine-Large Animal Clinical Sciences Department, Florida Cooperative Extension Service, Institute of Food and Agricultural Sciences, University of Florida, pp. 1–2.

Caffrey, N.P., Dohoo, I.R., Cockram, M.S., 2017. Factors affecting mortality risk during transportation of broiler chickens for slaughter in Atlantic Canada. Prev. Vet. Med. 147, 199–208.

Cahaner, A., Ajuh, J.A., Siegmund-Schultze, M., Azoulay, Y., Druyan, S., Zarate, A.V., 2008. Effects of the genetically reduced feather coverage in naked neck and featherless broilers on their performance under hot conditions. Poult. Sci. 87, 2517–2527.

Cirule, D., Tatjana, K., Jolanta, V., Markus, J.R., Krams, I., 2012. Rapid effect of handling on counts of white blood cells in a wintering passerine bird: a more practical measure of stress. J. Ornithol. 153, 161–166.

Cockrem, J.F., 2007. Stress, corticosterone responses and avian personalities. J. Ornithol. 148, 169–178.

Dadgar, S., Lee, E.S., Leer, T.L.V., Burlinguette, N., Classen, H.L., Crowe, T.G., Shand, P.J., 2010. Effect of microclimate temperature during transportation of broiler chickens on quality of the pectoralis major muscle. Poult. Sci. 89, 1033–1041.

Daghir, N.J., Lebanon, B., 2009. Nutritional strategies to reduce heat stress in broilers and broiler breeders. Loh. Inform. 44, 1–11.

Dai, S.F., Gao, F., Xu, X.L., Zhang, W.H., Song, S.X., Zhou, G.H., 2012. Effects of dietary glutamine and gamma-aminobutyric acid on meat colour, pH, composition, and water-holding characteristic in broilers under cyclic heat stress. Br. Poult. Sci. 53, 471–481.

Dale, N.M., Fuller, H.L., 1979. Effects of diet composition on feed intake and growth of chicks under heat stressI. Dietary fat levels. Poult. Sci. 58, 1529–1534.

Dantzer, R., Keith, W.K., 1989. Stress and immunity: an integrated view of relationships between the brain and the immune system. Life Sci. 44, 1995–2008.

Darszon, A., Labarca, P., Nishigaki, T., Espinosa, F., 1999. Ion channels in sperm. Physiol. Rev. 79, 481–510.

De Passille, A.M., Rushen, J., 2005. Food safety and environmental issues in animal welfare. Rev. Sci. Tech. Off. Int. Epiz. 24, 757–766.

De Zoete, M.R., Keestra, A.M., Roszczenko, P., Van Putten, J.P.M., 2010. Activation of human and chicken toll-Like receptors by Campylobacter spp. Infect. Immun. 78, 1229–1238.

Decuypere, E., Buyse, J., Mérat, P., Zoons, J., Vloeberghs, J., 1993. Growth, abdominal fat content, heat production and plasma hormone levels of naked-neck and control broiler chickens. Anim. Prod. 57, 483–490.

Deeb, J., Cahaner, A., 2001. Genotype-by-environment interaction with broiler genotypes differing in growth rate: 2. The effects of high ambient temperature on dwarf versus normal broilers. Poult. Sci. 80, 541–548.

Deeb, J., Cahaner, A., 2002. Genotype-by-environment interaction with broiler genotypes differing in growth rate. 3. Growth rate and water consumption of broiler progeny from weight-selected versus nonselected parents under normal and high ambient temperatures. Poult. Sci. 81, 293–301.

Deeb, J., Shlosberg, A., Cahaner, A., 2002. Genotype-by-environment interaction with broiler genotypes differing in growth rate. 4. association between responses to heat stress and to cold-induced ascites. Poult. Sci. 81, 1454–1462.

Dickens, M.J., Delehanty, D.J., Romero, L.M., 2010. Stress: an inevitable component of animal translocation. Biol. Conserv. 143, 1329–1341.

Ebeid, T.A., Suzuki, T., Sugiyama, T., 2012. High ambient temperature influences eggshell quality and Calbindin-D28k localization of eggshell gland and all intestinal segments of laying Hens. Poult. Sci. 91, 2282–2287.

El-Tarabany, M.S., 2016. Effect of thermal stress on fertility and egg quality of Japanese quail. J. Therm. Biol. 61, 38–43.

Etches, R.J., John, T.M., Verrinder-Gibbins, A.M., 2008. Behavioural, physiological, neuroendocrine and molecular responses to heat stress. In: Book Poultry Production in Hot Climates. CABI, pp. 48.

Fan, L., Xu, Y.M., Qin, Y.Q., Yuan, T.L., Heng, S., Jing, F., 2015. PHD3 stabilizes the tight junction protein occludin and protects intestinal epithelial barrier function. J. Biol. Chem. 290, 2058–2089.

Fisinin, V.I., Kavtarashvili, A.S., 2015. Heat stress in poultry. II. Methods and techniques for prevention and alleviation. Agric. Biol. 50, 431–443.

Gallo, A., Tosti, E., 2015. Ion currents involved in gamete physiology. Int. J. Dev. Biol. 59, 261–270.

Ghazalah, A.A., Elsamee, M.O.A., Ali, A.M., 2008. Influence of dietary energy and poultry fat on the response of broiler chicks to heat. Therm. Int. J. Poult. Sci. 7, 355–359.

Ghazi, S., Habibian, M., Moeini, M.M., Abdol-mohammadi, A.R., 2012. Effects of different levels of organic and inorganic chromium on growth performance and Immunocompetence of broilers under heat stress. Biol. Trac. Elem. Res. 146, 309–317.

Gomes, A.V.S., Quinteiro-Filho, W.M., Ribeiro, A., Ferraz-de-Paula, V., Pinheiro, M.L., Baskeville, E., Akamine, A.T., Astolfi-Ferreira, C.S., Ferreira, A.J.P., Palermo-Neto, J., 2014. Overcrowding stress decreases macrophage activity and increases salmonella enteritidis invasion in broiler chickens overcrowding stress decreases macrophage activity and increases salmonella enteritidis invasion in broiler chickens. Avian Pathol. 43, 82–90.

Goulet, O., 2015. Potential role of the intestinal microbiota in programming health and disease. Nut. Revi 73, 32–40.

Gupta, S.K., Shinde, K.P., Lone, S.A., 2016. The potential impact of heat stress on production and reproduction of dairy animals: consequences and possible solutions: a review. Int. J. Sci. Environ. Technol. 5, 903–911.

Guy, J.H., Vincent, G.E., 2018. Nutrition and supplementation considerations to limit endotoxemia when exercising in the heat. Sports 6, 1–12.

Gwaza, D., Nachi, D., 2015. Effect of naked neck gene on egg and body weight of chickens on free range in selected Nigerian local chicken populations. J. Agric. Vet. Sci. 8, 119–122.

Gwaza, D.S., Ochefu, J., Victor, G., 2017. Strain by environmental interaction effects on broiler chickens performance in the derived Southern Guinea Savannah region of Nigeria. J. Res. Rep. Genet. 1, 18–22.

Hoffmann, I., 2010. Climate change and the characterization, breeding and conservation of animal genetic resources. Anim. Genet. 41, 32–46.

Hu, Y.J., Wang, Y.D., Tan, F.Q., Yang, W.X., 2013. Regulation of paracellular permeability: factors and mechanisms. Mol. Biol. Rep. 40, 6123–6142.

Huang, S., 2017. Broiler chickens under acute heat stress. Braz. J. Poult. Sci. 19, 87–94.

Ibtisham, F., Zhang, L., Xiao, M., An, L., Bilal, M., Nawab, A., Zhao, Y., Li, G., Xu, Y.M., 2017a. Review article genomic selection and its application in animal breeding. Thai. J. Vet. Med. 47, 301–310.

Ibtisham, F., An, L., Li, T., Niu, Y., Xiao, M., Zhang, L., Jia, R., 2017b. Growth patterns of two Chinese native goose breeds. Braz. J. Poult. Sci. 19, 203–210.

Ibtisham, F., Nawab, A., Li, G., Xiao, M., An, L., Naseer, G., 2018. Effect of nutrition on reproductive efficiency of dairy animals. Med. Weter. 74, 356–361.

Jahejo, A.R., Leghari, I.H., Sethar, A., Rao, M.N., Nisa, M., Sethar, G.H., 2016. Effect of heat stress and ascorbic acid on gut morphology of broiler chicken. Sindh. Univ. Res. J. 48, 829–832.

John, M., 2009. Functional morphology of the avian respiratory system, the lung-air sac system: efficiency built on complexity. Ostrich 79, 117–132.

Kala, M., Shaikh, M.V., Nivsarkar, M., 2017. Equilibrium between antioxidants and reactive oxygen species: a requisite for oocyte development and maturation. Reprod. Med. Biol. 16, 28–35.

Kamada, N., Seo, S.U., Chen, G.Y., Núñez, G., 2013. Role of the gut microbiota in immunity and inflammatory disease. Nat. Rev. Immunol. 13, 321–335.

Kapetanov, M., Pajić, M., Ljubojević, D., Pelić, M.L., 2015. Heat stress in poultry industry. Arh. Vet. Med. 8, 87–101.

Karnati, H.K., Pasupuleti, S.R., Kandi, R., Undi, R.B., Sahu, I., Kannaki, T.R., Subbiah, M., Gutti, R.K., 2015. TLR-4 signalling pathway: MyD88 independent pathway up-regulation in chicken breeds upon LPS treatment. Vet. Res. Commun. 39, 73–78.

Katarzyna, O., Sembratowicz, I., 2012. Stress as a factor modifying the metabolism in poultry. A review. Ann. Zootech. 30, 34–43.

Khajavi, M., Rahimi, S., Hassan, Z.M., Kamali, M.A., Mousavi, T., 2003. Effect of feed restriction early in life on humoral and cellular immunity of two commercial broiler strains under heat stress conditions. Br. Poult. Sci. 44, 490–497.

Kim, H.W., Cramer, T., Ogbeifun, O.O.E., Seo, J.K., Yan, F., Cheng, H.W., Kim, Y.H.B., 2017. meat and muscle biology breast meat quality and protein functionality of

broilers with different probiotic levels and cyclic heat challenge exposure. Meat Muscl. Biol. 1, 81–89.
Kong, L.N., Zhang, D.X., Ji, C.L., Zhang, X.Q., Luo, Q.B., 2015. Association analysis between SNPs in the 5′- flanking region of the chicken GRP78 Gene, thermotolerance parameters, and tissue mRNA expression. Genet. Mol. Res. 14, 6110–6123.
Kumari, K., Raja, N., Nath, D.N., 2018. Ameliorative measures to counter heat stress in poultry. World Poult. Sci. J. 74, 117–130.
Lan, P.T.N., Sakamoto, M., Benno, Y., 2004. Effects of two probiotic lactobacillus strains on Jejunal and Cecal microbiota of broiler chicken under acute heat stress condition as revealed by molecular analysis of 16S rRNA genes. Microbiol. Immunol. 48, 917–929.
Lara, L.J., Rostagno, M.R., 2013. Impact of heat stress on poultry production. Animals 3, 356–369.
Leinonen, I., Williams, A.G., Kyriazakis, I., 2014. The effects of welfare-enhancing system changes on the environmental impacts of broiler and egg production. Poult. Sci. 93, 256–266.
Lin, H., Wang, L.F., Song, J.L., Xie, Y.M., Yang, Q.M., 2002. Effect of dietary supplemental levels of vitamin A on the egg production and immune responses of heat-stressed laying hens. Poult. Sci. 81, 458–465.
Lin, H., Jiao, H.C., Buyse, J., Decuypere, E., 2006. Strategies for preventing heat stress in poultry. World Poult. Sci. J. 62, 71–86.
Lotvedt, P., Fallahshahroudi, A., Bektic, L., Altimiras, J., Jensen, P., 2017. Chicken domestication changes expression of stress-related genes in brain, pituitary and adrenals. Neurobiol. Stress. 7, 113–121.
Loyau, T., Berri, C., Bedrani, L., Praud, C., Duclos, M.J., 2013. Thermal manipulation of the embryo modifies the physiology and body composition of broiler chickens reared in floor pens without affecting breast meat processing quality. J. Anim. Sci. 91, 3674–3685.
Mack, L.A., Felver-Gant, J.N., Dennis, R.L., Cheng, H.W., 2013. Genetic variations alter production and behavioral responses following heat stress in 2 strains of laying hens. Poult. Sci. 92, 285–294.
Manuja, B., Manuja, A., Aich, P., 2012. Stress and its Impact on farm animals. Front. Biosci. E4, 1759–1767.
Mateos, G.G., Sell, J.L., Eastwood, J.A., 1982. Rate of food passage (transit time) as influenced by level of supplemental fat. Poult. Sci. 61, 94–100.
Mazzi, C.M., Ferro, J.A., Inês, M., Ferro, T., José, V., Savino, M., Augusto, A., Coelho, D., Macari, M., 2003. Polymorphism analysis of the hsp70 stress gene in broiler chickens (Gallus gallus) of different breeds. Genet. Mol. Biol. 26, 275–281.
Mideksa, B.A., 2017. Opportunities of preserving local animal genetic resources in Ethiopia. Glob. J. Anim. Breed. Genet. 5, 369–376.
Moberg, G.P., 2000. Biological response to stress: implications for animal welfare. biology of animal stress. In the Book of the Biology of Animal Stress: Basic Principles and Implications for Animal Welfare. pp. 1–21.
Mormede, P., Andanson, S., Auperin, B., Beerda, B., Guemene, D., Malmkvist, J., Manteca, X., 2007. Exploration of the hypothalamic-pituitary-adrenal function as a tool to evaluate animal welfare. Physiol. Behav. 92, 317–339.
Nazar, F.N., Estevez, I., Correa, S.G., Marin, R.H., 2017. Stress induced polarization of immune-neuroendocrine phenotypes in Gallus Gallus. Sci. Report. 7, 1–11.
Neves, D.P., Naas, I.A., Vercellino, R.A., Moura, D.J., 2010. Do broilers prefer to eat from a certain type of feeder? Braz. J. Poult. Sci. 12, 179–187.
Neves, D.P., Banhazi, T.M., Naas, I.A., 2014. Feeding behaviour of broiler chickens: a review on the biomechanical characteristics. Braz. J. Poult. Sci. 16, 1–16.
Nienabar, J.A., 2007. Livestock production system management responses to thermal challenges. Int. J. Biometeorol. 52, 149–157.
Noiva, R.M., Menezes, A.C., Peleteiro, M.C., 2014. Influence of temperature and humidity manipulation on chicken embryonic development. Vet. Res. 10, 2–10.
Papadopoulou, A., Petrotos, K., Stagos, D., Gerasopoulos, K., Maimaris, A., Makris, H., Kafantaris, I., 2017. Enhancement of antioxidant mechanisms and reduction of oxidative stress in chickens after the administration of drinking water enriched with polyphenolic powder from olive mill waste waters. Oxidat. Med. Cellul. Long. 2017, 1–11.
Patra, B.N., Bais, R.K.S., Prasad, R.B., Singh, B.P., 2002. Performance of naked neck versus normally feathered coloured broilers for growth, carcass traits and blood biochemical parameters in tropical climate. Asian-Aust. J. Anim. Sci. 12, 1776–1783.
Pawar, S.S., Basavaraj, S., Dhansing, L.V., Nitin, K.P., Sahebrao, K.A., Vitthal, N.A., Manoj, B.P., Kumar, B.S., 2016. Assessing and mitigating the impact of heat stress in poultry. Adv. Anim. Vet. Sci. 4, 332–341.
Perez-Moreno, M., Fuchs, E., 2006. Catenins: keeping cells from getting their signals crossed. Dev. Cell. 11, 601–612.
Prieto, M.T., Campo, J.L., 2010. Effect of heat and several additives related to stress levels on fluctuating asymmetry, heterophil: lymphocyte ratio, and tonic immobility duration in White Leghorn Chicks. Poult. Sci. 89, 2071–2077.
Quinteiro-Filho, W.M., Gomes, A.V.S., Pinheiro, M.L., Ribeiro, A., Ferraz-de-Paula, V., Astolfi-Ferreira, C.S., Ferreira, A.J.P., Palermo-Neto, J., 2012. Heat stress impairs performance and induces intestinal inflammation in broiler chickens infected with Salmonella Enteritidis. Avian Pathol. 41, 421–427.
Rahman, M.S., Pramanik, M.A.H., Basak, B., Tarafdar, S.U., Biswas, S.K., 2002. Effect of feeding low protein diets on the performance of broilers during hot-humid season. Int. J. Poult. Sci. 1, 35–39.
Raju, M.V.L.N., Sunder, G.S., Chawak, M.M., Rao, S.V.R., Sadagopan, V.R., 2004. Response of naked neck (Nana) and normal (Nana) broiler chickens to dietary energy levels in a subtropical climate. Br. Poult. Sci. 45, 186–193.
Renaudeau, D., Collin, A., Yahav, S., De Basilio, V., Gourdine, J.L., Collier, R., 2012. Adaptation to hot climate and strategies to alleviate heat stress in livestock production. Animal 6, 707–728.
Riaz, A., Aleem, M., Ijaz, A., Saeed, M.A., Latif, A., 2004. Effect of collection frequency on the semen quality of broiler breeder. Br. Poult. Sci. 45, 823–827.
Romero, L.M., Platts, S.H., Schoech, S.J., Wada, H., Crespi, E., Martin, L.B., Buck, C.L., 2015. Understanding stress in the healthy animal–potential paths for progress. Stress 18, 491–497.
Sahin, K., Sahin, N., Kucuk, O., Hayirli, A., Prasad, A.S., 2009. Role of dietary zinc in heat-stressed poultry: a review. Poult. Sci. 88, 2176–2183.
Salvo-romero, E., Carmen, A.C., Cristina, P.C., Maite, C.B., María, V., 2015. The intestinal barrier function and its involvement in digestive disease. Revesp. Enferm. Dig. 107, 686–695.
Santi, C.M., Orta, G., Salkoff, L., Visconti, P.E., Darszon, A., Treviño, C.L., 2013. K(+) and Cl(−) channels and transporters in sperm function. Curr. Top. Dev. Biol. 102, 385–421.
Scholtz, M.M., Van Zyl, J.P., Theunissen, A., 2014. The effect of epigenetic changes on animal production. Appl. Anim. Husb. Rur. Dev. 7, 7–10.
Sebho, H.K., 2016. Exotic chicken status, production performance and constraints in ethiopia: a review. Asian J. Poult. Sci. 10, 30–39.
Settar, P., Yalcin, S., Turkmut, L., Ozkan, S., Cahanar, A., 1999. Season by genotype interaction related to broiler growth rate and heat tolerance. Poult. Sci. 78, 1353–1358.
Shini, S., Kaiser, P., Shini, A., Bryden, W.L., 2008. Biological response of chickens (Gallus Gallus Domesticus) induced by corticosterone and a bacterial endotoxin. Comp. Biochem. Physiol. Part B Biochem. Mol. Biol. 149, 324–333.
Shini, S., Huff, G.R., Shini, A., Kaiser, P., 2010. Understanding stress-induced immunosuppression: exploration of cytokine and chemokine gene profiles in chicken peripheral leukocytes. Poult. Sci. 89, 841–851.
Siddiqui, M.R., Mayanil, C.S., Kim, K.S., Tomita, T., 2015. Angiopoietin-1 regulates brain endothelial permeability through PTPN-2 mediated tyrosine dephosphorylation of occludin. PLoS One 10, 1–13.
Siegel, H.S., 1980. Physiological stress in birds. BioScience 30, 529–534.
Smith, S.M., Vale, W.W., 2006. The role of the hypothalamic-pituitary-adrenal axis in neuroendocrine responses to stress. Dialog. Clin. Neurosci. 8, 383–395.
Sohail, M.U., Hume, M.E., Byrd, J.A., Nisbet, D.J., Ijaz, A., Sohail, A., Shabbir, M.Z., Rehman, H., 2012. Effect of supplementation of prebiotic mannan-oligosaccharides and probiotic mixture on growth performance of broilers subjected to chronic heat stress. Poult. Sci. 91, 2235–2240.
Sokołowicz, Z., Krawczyk, J., Świątkiewicz, S., 2016. Quality of poultry meat from native chicken breeds – a review. Ann. Anim. Sci. 16, 347–368.
Sugiharto, S., Yudiarti, T., Isroli, I., Widiastuti, E., Kusumanti, E., 2017. Dietary supplementation of probiotics in poultry exposed to heat stress – a review. Ann. Anim. Sci. 17, 591–604.
Tellez, J.G., Tellez-isaias, G., Dridi, S., 2017. Heat stress and gut health in broilers: role of tight junction proteins. Adv. Food Technol. Nutr. Sci. 3, 1–4.
Tirawattanawanich, C., Chantakru, S., Nimitsantiwong, W., Tongyai, S., 2011. The effects of tropical environmental conditions on the stress and immune responses of commercial broilers, thai indigenous chickens, and crossbred chickens. Poult. Sci. 20, 409–420.
Triantaphyllopoulos, K.A., Ikonomopoulos, I., Bannister, A.J., 2016. Epigenetics and inheritance of phenotype variation in livestock. Epigen. Chromat. 9, 1–18.
Tuohy, K.M., Probert, H.M., Smejkal, C.W., Gibson, G.R., 2003. Using probiotics and prebiotics to improve gut health. Drug. Dis. Tod. 8, 692–700.
Varasteh, S., Braber, S., Akbari, P., Garssen, J., Johanna, F.G., 2015. Differences in susceptibility to heat stress along the chicken intestine and the protective effects of Galacto-Oligosaccharides. PLoS One 10, 1–18.
Vecerek, V., Voslarova, E., Conte, F., Vecerkova, L., Bedanova, I., 2016. Negative trends in transport-related mortality rates in broiler chickens. Asian-Aus. J. Anim. Sci. 29, 1796–1804.
Virden, W.S., Kidd, M.T., 2009. Physiological stress in broilers: ramifications on nutrient digestibility and responses. J. Appl. Poult. Res. 18, 338–347.
Vos, Rob, 2015. Thought for Food: Strengthening Global Governance of Food Security 29. United Nations (Department of Economic and Social Affairs), New York, USA, pp. 1–21.
Yalcin, S., Testik, A., Ozkan, S., Settar, P., Cahaner, A., 1997. Performance of naked neck and normal broilers in hot, warm, and temperate climates genetic stock and management climate in poultry houses. Poult. Sci. 76, 930–937.
Yosi, F., Widjastuti, T., Setiyatwan, H., 2017. Performance and physiological responses of broiler chickens supplemented with potassium chloride in drinking water Under environmental heat stress. Asian. J. Poult. Sci. 11, 31–37.
Yu, J., Bao, E., Yan, J., Lei, L., 2008. Expression and localization of Hsps in the heart and blood vessel of heat-stressed broilers. Cell Stress Chapter 13, 327–335.
Yunis, R., Cahaner, A., 1999. The effects of the Naked Neck (Na) and Frizzle (F) genes on growth and meat yield of broilers and their interactions with ambient temperatures and potential growth rate. Poult. Sci. 78, 1347–1352.
Zerjal, T., Gourichon, D., Rivet, B., Bordas, A., 2013. Performance comparison of laying hens segregating for the frizzle gene under thermoneutral and high ambient temperatures. Poult. Sci. 92, 1474–1485.
Zhang, P., Yan, T., Wang, X., Kuang, S., Xiao, Y., Lu, W., Bi, D., 2017. Probiotic mixture ameliorates heat stress of laying hens by enhancing intestinal barrier function and improving gut microbiota. Ital. J. Anim. Sci. 16, 292–300.
Zoetendal, E.G., Collier, C.T., Koike, S., Mackie, R.I., Gaskins, H.R., 2004. Molecular ecological analysis of the gastrointestinal microbiota: a review. J. Nutr. 134, 465–472.
Zulkifli, I., Dunnington, E.A., Gross, W.B., Siegel, P.B., 1994. Inhibition of adrenal steroidogenesis, food restriction and acclimation to high ambient temperatures in chickens. Br. Poult. Sci. 35, 417–426.